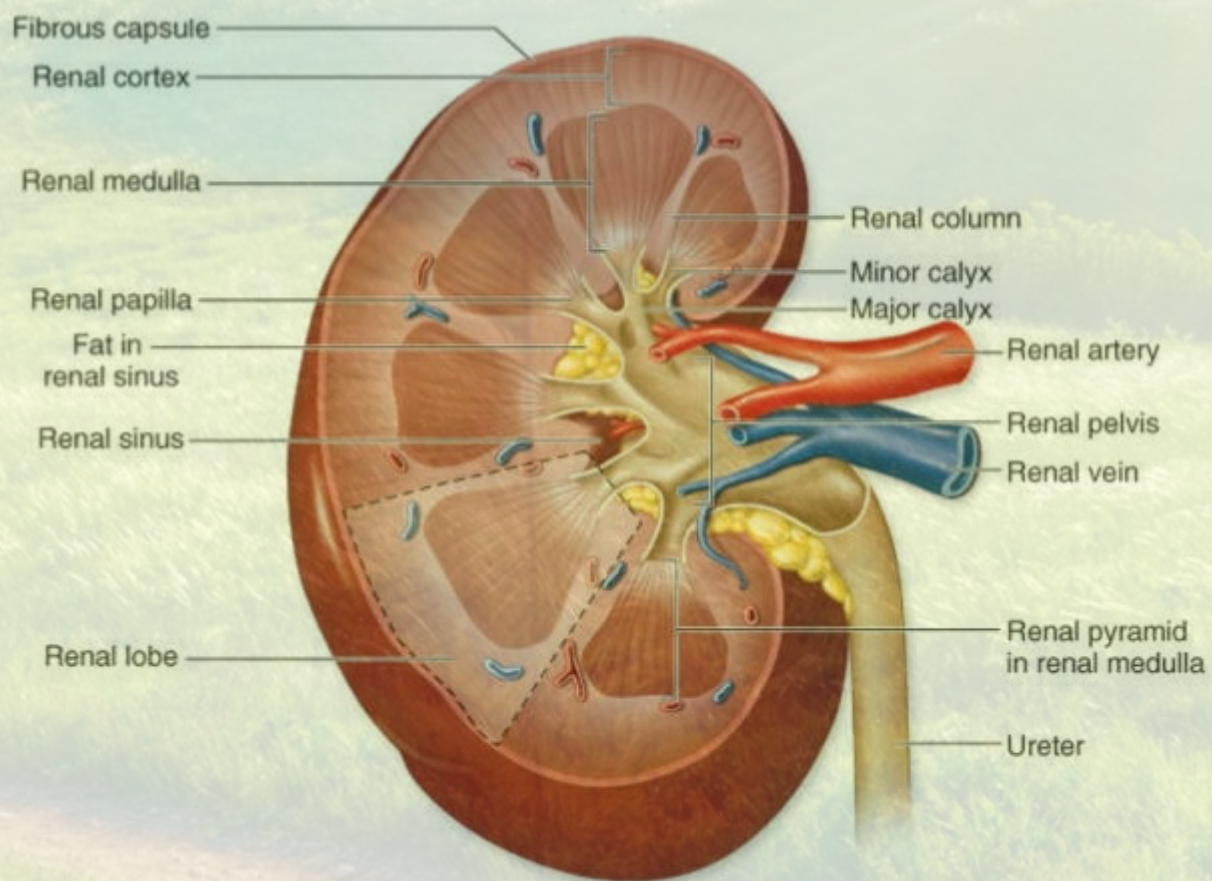


KIDNEY



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GLOMERULONEPHRITIS (GN)

DEFINITION

- Renal diseases characterized by GLOMERULAR INJURY & INFLAMMATION:

- Mainly immunologically mediated.
- Bilateral & Symmetrical affection of the kidneys.

ETIOLOGY

I. PRIMARY

(Idiopathic & pathology is confined to kidney)

II. SECONDARY

(a part of multisystem disorder)

1. Infections:

- Bacterial: Post – Streptococcal, SBE, Syphilis.
- Viral: HBV, HCV, HIV.
- Parasitic: Schistosoma.

2. Immune:

SLE, PAN, HSP, GPS, EMC.

3. Iatrogenic:

Gold, Penicillamine, Captopril.

4. METABOLIC:

DM, Amyloidosis.

5. MALIGNANCY:

MM, Lymphoma.

6. MISCELLANEOUS:

- Hereditary: Alport's syndrome, Sickle cell disease.
- MAHA: TTP, HUS, DIC.
- Hypertensive emergency: Malignant hypertension.
- Toxic: Heroin, Mercury.

PATHOGENESIS

VIP

1. IMMUNOLOGICAL INJURY:

- Immune complexes: *most* types of GN, e.g. PSGN, SBE, SLE.
- Anti – GBM antibodies: *some* types of GN, e.g. GPS.
- Abnormal complement activation: *some cases of idiopathic GN*.

2. NON – IMMUNOLOGICAL INJURY:

- A. Accumulation of abnormal metabolites: e.g.
 - DM.
 - Amyloidosis.
- B. High intraglomerular pressure: e.g.
 - Hypertensive emergency: *Malignant hypertension*.
- C. Intravascular coagulation: e.g.
 - TTP, HUS, DIC.
- D. Deposition diseases: e.g.
 - EMC.
 - Amyloidosis.
 - MM.
 - *In these diseases, abnormal proteins are deposited in the glomeruli →
→ inflammatory reaction and / or glomerulosclerosis.*
- E. Toxic injury:
 - Heroin, Mercury.
- F. INFECTION:
 - Direct infection of renal cells.
 - Release of nephrotoxins: e.g. E – coli → HUS.
 - Immune complexes: e.g. PSGN, SBE.
 - Chronic stimulation of amyloid formation.

CLASSIFICATION

1. Etiology:

[of glomerular injury]

- Primary: idiopathic.
- Secondary: a part of multisystem disorder.

2. Course:

[of glomerular injury]

- Acute: over days to weeks.
- Subacute (*rapidly progressive*): over weeks to months.
- Chronic: over months to years.

3. Distribution:

[of glomerular injury]

a) In the kidney:

- Diffuse: *Majority* of glomeruli (> 50 %).
- Focal: *Minority* of glomeruli (< 50 %).

b) In the glomerulus:

- Global: affects *almost all* of the glomerulus.
- Segmental: affects *only some parts* of the glomerulus.

4. Pathology:

[of glomerular injury]

a) Non – Proliferative = “no increase in glomerular cell number”

Non – Proliferative GN

1. Diffuse: *Minimal change GN, Membranous GN.*
2. Focal: *Focal segmental glomerulosclerosis.*

b) Proliferative = “increase in glomerular cell number” ... Why ??

1. Infiltration of: glomerulus by inflammatory cells (neutrophils & monocytes).
2. Proliferation of: resident glomerular cells:
 - Intracapillary proliferation: *endothelial or mesangial cells.*
 - Extracapillary proliferation: *epithelial cells in Bowman’s space.*

Proliferative GN

1. Diffuse:
 - *Membrano – proliferative GN.*
 - *Mesangio – proliferative GN.*
 - *Crescentic GN.*
 - *Diffuse – proliferative GN.*
2. Focal: *IgA nephropathy (Berger’s disease).*

PATHOLOGY

I. Non – Proliferative GN:

1. Diffuse:

A) Minimal change GN:

- Light microscope: No abnormality.
- Electron microscope: Diffuse EFFACEMENT of the foot processes of the PODOCYTES.
- It usually presents by: Nephrotic syndrome.
- It is the most common form of Nephrotic syndrome in: CHILDREN.

B) Membranous GN:

- There is: Diffuse LEAKY THICKENING of the GBM.
- It usually presents by: Nephrotic syndrome or asymptomatic proteinuria.
- It is the most common form of Nephrotic syndrome in: ADULTS.

2. Focal: “Focal segmental glomerulosclerosis”

- There is: sclerosis (fibrosis & scarring) & hyalinosis.
- Focal sclerosis: involves *Minority* of glomeruli (< 50 %).
- Segmental sclerosis: involves *only some parts* of the glomerulus.
- It usually presents by: Nephrotic syndrome.
- It causes Nephrotic syndrome in: CHILDREN & ADULTS.

II. Proliferative GN:

1. Diffuse:

A) Membrano – proliferative GN:

- There is: Diffuse LEAKY THICKENING of the GBM.
- There is: Intracapillary Proliferation
[Mesangial cells, Matrix & Endothelial cells].
- It usually presents by: Nephrotic syndrome or Nephritic syndrome.

B) **Mesangio – proliferative GN:**

- There is: Marked Proliferation of Mesangial cells and Matrix → encroachment on capillary lumen.
- It usually presents by: Nephritic syndrome.

C) **Crescentic GN (RPGN)**

- There is: Extracapillary Proliferation (marked & rapid) [Parietal epithelial cells in Bowman's space]
- There is: Marked Epithelial crescent formation →→→→
 - encroachment on capillary lumen
 - reduction of Bowman's space
- It usually presents by: Rapidly progressive RF (over weeks to months).

D) **Diffuse – proliferative GN:**

- There is: Intracapillary & Extracapillary Proliferation Endothelial, Mesangial & Epithelial cells.
- It usually presents by: Nephritic syndrome & ARF (over days to weeks)

2. **Focal:** “IgA nephropathy or Berger's disease”

- There is: mesangial proliferation, glomerular inflammation & necrosis.
- Focal affection: involves *Minority* of glomeruli (< 50 %).
- Segmental affection: involves *only some parts* of the glomerulus.
- It usually presents by: Hematuria, mild to moderate ↓GFR, Nephritic syndrome.
- It is the most common type of GN world – wide in: ADULTS.

Chronic GN

- It is a chronic form characterized by:
 - *Glomerular:* atrophy.
 - *Tubular:* atrophy.
 - *Interstitial:* fibrosis.
- It is the end – stage of other types of GN.
- It presents with: CRF & ESRD.

CLINICAL PRESENTATIONS

- GN may present with one or more of the following clinical presentations:

1. Nephrotic syndrome.
2. Nephritic syndrome (Acute GN).
3. Urinary abnormalities: e.g. *hematuria*, *proteinuria*.
4. ARF: (over days to weeks).
5. Rapidly progressive RF: (over weeks to months).
6. CRF: (over months to years).
7. HYPERTENSION.

INVESTIGATIONS

1. Urine analysis.

2. Blood investigations:

e.g.

- *CBC*
- *Lipid profile.*
- *Plasma proteins.*
- *Electrolytes.*

3. Renal function tests.

4. Renal imaging:

e.g.

- *Abdominal ultrasonography.*

5. Renal biopsy.

6. Investigations for the cause:

e.g.

- *Hepatitis markers:* for *HBV*, *HCV*.
- *Serology:* for *SLE*.
- *Blood glucose:* for *DM*.

NEPHRITIC SYNDROME

“ ACUTE GN ”

DEFINITION

- A disease characterized by the ACUTE ONSET of:

1. HEMATURIA.
2. PROTEINURIA (Sub – nephrotic).
3. OLIGURIA (& ARF).
4. OEDEMA.
5. HYPERTENSION.

ETIOLOGY

I. PRIMARY

(Idiopathic & pathology is confined to kidney)

II. SECONDARY

(a part of multisystem disorder)

1. Infections:

- Bacterial: Post – Streptococcal, SBE.
- Viral: HBV, HCV.

2. Immune:

SLE, PAN, HSP, GPS, EMC.

PATHOLOGY

“Proliferative GN”

1. Diffuse:

- A) Membrano – proliferative GN.
- B) Mesangio – proliferative GN.
- C) Diffuse – proliferative GN. ★

2. Focal:

“IgA nephropathy or Berger’s disease”.

ACUTE POST – STREPTOCOCCAL GN (PSGN)

ETIOLOGY

- Organism: *Nephritogenic strains of group A β - hemolytic streptococci.*
- Site of infection: Pharynx or skin.
- Incubation period: *1 – 3 weeks.*
- Age: *Young (usually below 20).*
- Sex: *Males > Females (2 : 1).*

PATHOGENESIS

- Immunological injury is due to:
 - Deposition of **IMMUNE COMPLEXES** in the glomeruli.

PATHOLOGY

1. Features of: Diffuse proliferative GN.
2. Electron microscope: SUBEPITHELIAL IMMUNE DEPOSITS “**HUMPS**”.

CLINICAL PICTURE

“ACUTE NEPHRITIC SYNDROME”

1. HEMATURIA.
2. PROTEINURIA (Sub – nephrotic).
3. OLIGURIA (& ARF).
4. OEDEMA.
5. HYPERTENSION.

1. HEMATURIA:

- PATHOGENESIS:
 - Injury to the glomerular capillary wall.
- CLINICAL FEATURES:
 - It occurs in the form of smoky or dark urine, HOWEVER, frank hematuria may occur.

2. PROTEINURIA: (Sub – nephrotic)

- PATHOGENESIS:
 - Injury to the glomerular capillary wall.
- CLINICAL FEATURES:
 - It is usually detected in urine analysis: (< 3.5 gm / 24 h).

3. OLIGURIA: (& ARF)

- PATHOGENESIS: [$\downarrow\downarrow$ GFR]
 - Obstruction of Glomerular capillary lumen by the ***proliferative lesion:***
 1. Infiltration of: glomerulus by inflammatory cells (neutrophils & monocytes).
 2. Proliferation of: resident glomerular cells.
 - Intra – renal VC due to ***local imbalances of VC & VD substances:***
 1. VC substances: Leukotrienes & PAF.
 2. VD substances: Nitric oxide & Prostacyclin.
- CLINICAL FEATURES:
 - Acute onset of oliguria (< 400 ml / day) & ARF: over days to weeks.

4. OEDEMA:

- PATHOGENESIS:
 - $\downarrow\downarrow$ GFR.
 - $\uparrow\uparrow$ reabsorption of: salt & water.
- CLINICAL FEATURES:
 - Onset: Acute.
 - Site: Peri-orbital region & face (early), may become generalised (late).

5. HYPERTENSION:

○ PATHOGENESIS:

- ↓↓ GFR.
- ↑↑ reabsorption of: salt & water.
- Generalised VC: due to stimulation of RAAS.

○ CLINICAL FEATURES:

- Severe Hypertension may pass to: Hypertensive encephalopathy.
- Associated CVS manifestations: [due to hypervolemia]
Heart failure: may pass to Acute pulmonary oedema.

6. GENERAL MANIFESTATIONS: "3 P"

- **P**yrexia: + headache, malaise, anorexia, nausea, vomiting.
- **P**ain: in both loins.
- **P**allor: due to anemia.

INVESTIGATIONS

1. Urinalysis:

- *Volume:* oliguria (< 400 ml / day).
- *Aspect:* smoky or dark urine, However frank hematuria may occur.
- *Specific gravity:* increased.
- *Proteins:* proteinuria (Sub – nephritic < 3.5 gm / 24 h).

• MICROSCOPIC EXAMINATION:

- Cells: **Red B Cs** (many & dysmorphic)
- Casts: **Red** cell casts

The most important investigation

2. Blood investigations:

- *CBC:* Anemia & high ESR.
- *Lipid profile:* Normal.
- *Plasma proteins:* Normal.
- *Electrolytes:* ↑ K (may be present).

3. Renal function tests:

- *Impaired glomerular function:* ↓ creatinine clearance (a good estimate of GFR).
- *Blood urea & serum creatinine:* may be elevated.

4. Renal imaging:

- *Abdominal ultrasonography:* mild swelling of the kidneys.

5. Renal biopsy:

- *It is usually not needed for the diagnosis.*

6. Investigations for the cause:

- *Streptococcal antibody tests:* High ASO titre, Positive ASZ test.

PROGNOSIS

“Better in children than in adults”

- Over 90 % of cases: Complete recovery usually in few weeks.
- About 5 % of cases: RPGN → renal failure in weeks to months.
- Few cases: Death from ARF, Hypertensive encephalopathy, APE.

DIFFERENTIAL DIAGNOSIS

- From causes of:
 - *Nephritic syndrome.*
 - *Hematuria.*
 - *Proteinuria.*
 - *Oliguria & ARF.*
 - *Oedema.*
 - *Secondary hypertension.*
- From: Nephrotic syndrome.

TREATMENT

A. GENERAL MEASURES:

1. Rest: *Complete bed rest.*

2. Diet:

- ***Salt:*** restriction.
- ***Fluid:*** restriction in marked oliguria & oedema, allowed when diuresis starts.
- ***Potassium:*** restriction in case of hyperkalemia.
- ***Protein:*** restriction in case of renal failure.
- ***Fats:*** restriction is preferred because they are nauseating.
- ***CHO:*** no restriction.

B. SPECIFIC MEASURES:

1. Antibiotics: *Crystalline penicillin, 1 million U / 6 hours (1 week).*

2. Symptomatic ttt: **e.g.**

- Hypertension: *Anti – hypertensive drugs,* **e.g.** Hydralazine.
- Oedema: *Diuretics,* **e.g.** Frusemide.

3. TTT of complications: **e.g.**

- ARF.
- Hypertensive encephalopathy.
- Acute pulmonary oedema.

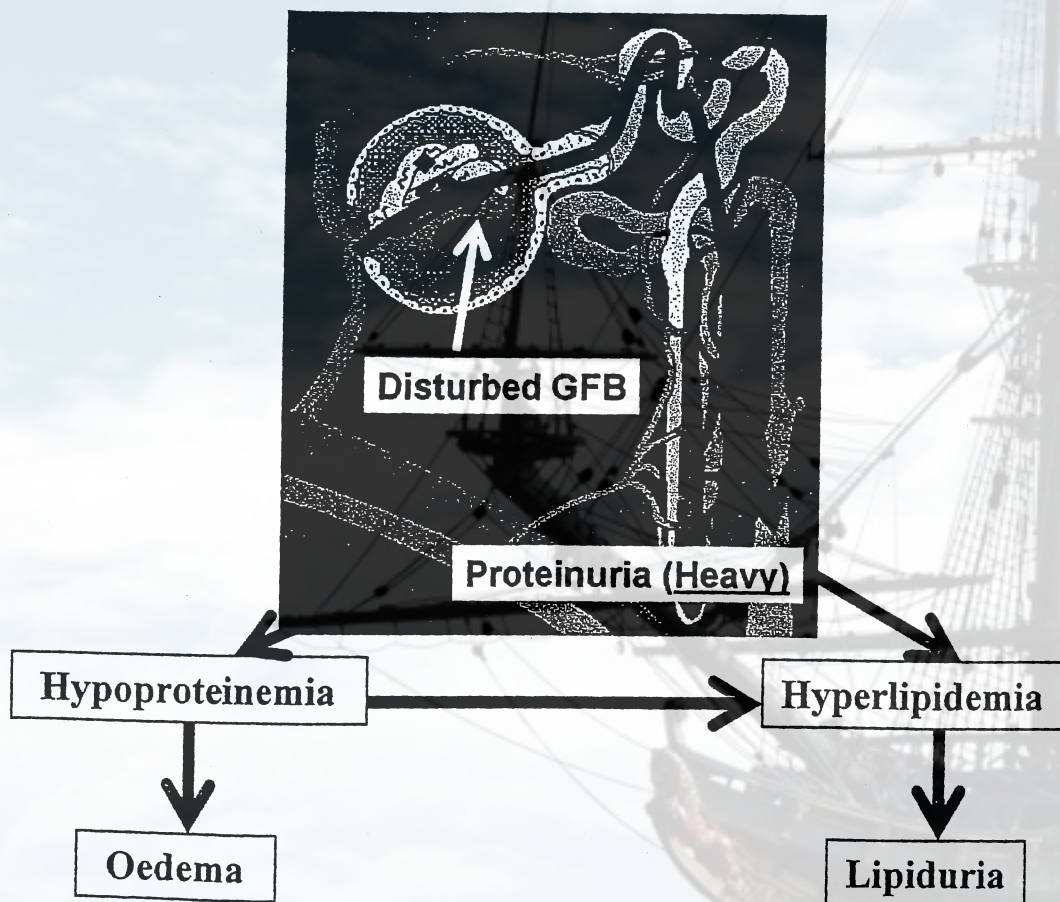
NEPHROTIC SYNDROME

DEFINITION

- A clinical syndrome characterized by:

1. PROTEINURIA (Heavy) (nephrotic > 3.5 gm / 24 h).
2. HYPOPROTEINEMIA.
3. OEDEMA.
4. HYPERLIPIDEMIA.
5. LIPIDURIA.

- Heavy proteinuria: is the primary abnormality.
- Other features: occur secondary to heavy proteinuria.



ETIOLOGY

I. PRIMARY

(Idiopathic & pathology is confined to kidney)

II. SECONDARY

(a part of multisystem disorder)

1. Infections:

- *Bacterial:* SBE, Syphilis.
- *Viral:* HBV HCV, HIV.
- *Parasitic:* Schistosoma.

2. Immune:

SLE, PAN, HSP, EMC.

3. Iatrogenic:

Gold, Penicillamine, Captopril.

4. METABOLIC:

DM, Amyloidosis.

5. MALIGNANCY:

MM, Lymphoma (Hodgkin's).

6. MISCELLANEOUS:

- *Hereditary:* Alport's syndrome, Sickle cell disease.
- *Toxic:* Heroin.

PATHOLOGY

I. Non – Proliferative GN:

1. Diffuse:

A) **Minimal change GN:** *The most common cause in* CHILDREN

B) **Membranous GN:** *The most common cause in:* ADULTS

2. Focal:

“Focal segmental glomerulosclerosis”

- It causes Nephrotic syndrome in: CHILDREN & ADULTS.

II. Proliferative GN:

- **Membrano – proliferative GN.**

CLINICAL PICTURE

1. PROTEINURIA (Heavy) (Nephrotic):

○ PATHOGENESIS:

- Leak of proteins through a disturbed GFB.....

○ CLINICAL FEATURES:

- It may be one of 2 types:

a) Selective proteinuria:

- Urine: proteins of low MW ONLY, e.g. albumin.
- Renal pathology: mild.
- Prognosis: good.

b) Non-selective proteinuria:

- Urine: proteins of low & high MW.
- Renal pathology: marked.
- Prognosis: poor.

- Loss of protein in urine will lead to:

a) Hypoproteinemia.

b) Tubular damage: which may progress to CRF.

2. HYPOPROTEINEMIA:

○ PATHOGENESIS:

- Heavy proteinuria.

○ CLINICAL FEATURES:

Loss of:

- Albumin → Generalized oedema.
- Immunoglobulins → Increased susceptibility to infections.
- Vit. D-binding globulin → Osteomalacia.
- Transferrin → Microcytic hypochromic anemia (*iron resistant*).
- Antithrombin – III → Hypercoagulability & Thromboembolism.

3. OEDEMA:

○ PATHOGENESIS:

- Hypoalbuminemia: ↓ oncotic pressure, *the most important factor*.
- Underfill Theory: Hypovolemia stimulates RAAS & stimulates ↑ ADH.

○ CLINICAL FEATURES:

- Onset: Gradual.
- Site: Peri-orbital region & face (early), may become generalised (late).
- Course: Generalized anasarca with ascites, pleural effusion, pericardial effusion.
- Character: Soft, pitting.

4. HYPERLIPIDEMIA “Increased cholesterol & triglycerides”

○ PATHOGENESIS:

- ↑ hepatic synthesis of lipids.
- ↓ removal of lipids.

○ CLINICAL FEATURES:

- Complications of: *ATHEROSCLEROSIS*.
- Xanthomata.

5. LIPIDURIA

- In urine, excess lipids & lipoproteins will pass as:
 - *Free fat*.
 - *Fatty casts (degenerated tubular epithelial cells containing fats)*.

6. Hypokalemia:

○ PATHOGENESIS:

- ↓ intake: Anorexia, nausea, vomiting.
- ↓ absorption: Oedema of the intestinal mucosa.
- ↑ aldosterone: Secondary hyperaldosteronism.
- DRUGS: Corticosteroids, Diuretics.

○ CLINICAL FEATURES:

- Refer to: “Hypokalemia”.

7. Hypocalcemia:

○ PATHOGENESIS:

- Loss of Albumin: will ↓ the albumin-bound Ca carried by this albumin.
- Loss of Vitamin D-binding *globulin*.

○ CLINICAL FEATURES:

- It RARELY results in TETANY because the Ionizable Ca is normal.

8. Other manifestations:

1. Pallor: due to anemia.
2. GIT: Anorexia, N, V, Malabsorption, due to oedema of the GIT.
3. Renal: Progression to: (CRF & secondary HTN) or (ARF).
 - CRF: due to progressive tubular damage by proteinuria.
 - ARF: due to severe hypovolemia or renal vein thrombosis.
4. CVS: Accelerated CVD (e.g. CAD & Stroke), due to:
 - Hyperlipidemia.
 - Hypercoagulability.

↑ Atherosclerosis

9. Manifestations of the cause:

- e.g. SLE, DM.

INVESTIGATIONS

1. Urinalysis:

- *Volume*: normal.
- *Aspect*: frothy.
- *Specific gravity*: increased
- *Proteins*: heavy proteinuria (nephrotic > 3.5 gm / 24 h).
Proteinuria may be: selective or non-selective.
- MICROSCOPIC EXAMINATION:
 - Casts: **Fatty** casts.

Blood investigations:

- *CBC:* Anemia.
- *Lipid profile:* ↑ cholesterol & triglycerides.
- *Plasma proteins:* ↓ (mainly, albumin).
- *Electrolytes:* ↓ K & ↓ Decreased total Ca.

Renal function tests:

- *Normal:* except if renal failure occurs.

Renal biopsy:

- *It will show the pathology.*

Investigations for the cause:

- *e.g.* SLE, DM.

PROGNOSIS

“It depends on several factors”

- *Pathology:* it is best in minimal change GN.
- *Age:* it is better in children than in adults.
- *Type of proteinuria:* it is better in selective than non-selective proteinuria.

DIFFERENTIAL DIAGNOSIS

- From causes of:
 - *Nephrotic syndrome.*
 - *Proteinuria.*
 - *Oedema.*
 - *Hyperlipidemia.*
- From: *Nephritic syndrome.*
- From: *Myxoedema.*

TREATMENT

A. TTT of the cause:

1. PRIMARY

“according to the pathology”

- Minimal change GN:

- Prednisone:

- 2 mg / Kg / day: for 4 weeks, then:

- 1 mg / Kg / day: for 4 weeks, then:

- Gradual withdrawal: over 4 weeks.

- Immunosuppressive drugs: “Add Cyclophosphamide”

- 2 mg / Kg / day: for 8 weeks.

- In corticosteroid – resistant cases.

- Membranous GN:

- FSGS:

- Membranoproliferative GN:

Corticosteroids
+
Immunosuppressive drugs

2. SECONDARY:

- e.g. *SLE, DM.*

B. SYMPTOMATIC TTT:

1. Hypoproteinemia:

- DIET: High protein.

- ALBUMIN: IV albumin.

2. Oedema:

- DIET: High protein + salt restriction.

- DIURETICS:

- K – sparing : spironolactone.

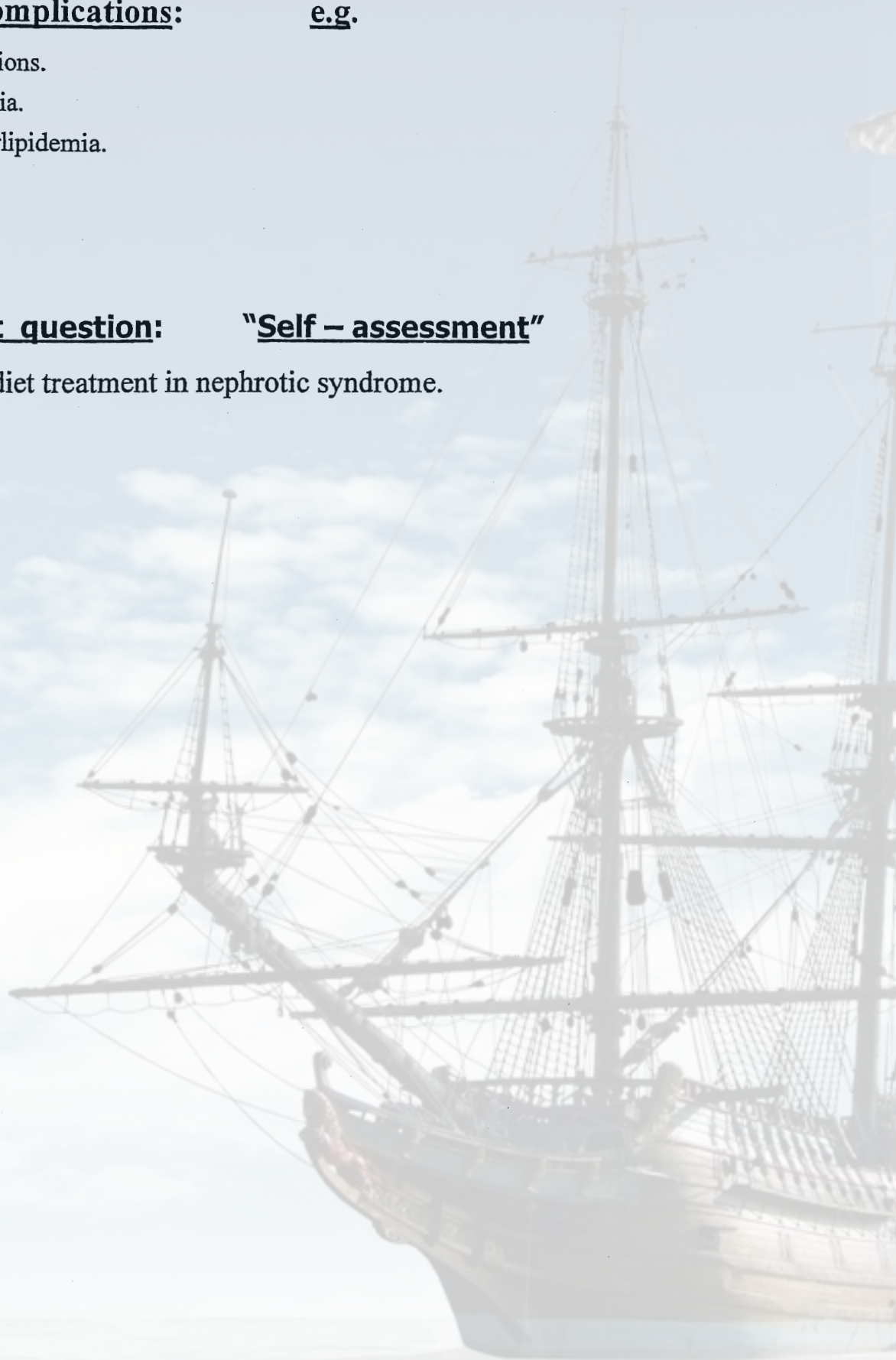
- Non K – sparing: furosemide or thiazides (add K).

3. **TTT of complications:** **e.g.**

- Infections.
- Anemia.
- Hyperlipidemia.

- **Important question:** **"Self – assessment"**

- Outline diet treatment in nephrotic syndrome.



INTERSTITIAL NEPHRITIS

DEFINITION

- A group of disorders that affect the renal interstitium & the renal tubules.

ETIOLOGY

I. Acute interstitial nephritis: “3 I”

1. Iatrogenic: [Allergic, drug hypersensitivity]
 - NSAIDs.
 - Antibiotics: *Methicillin, Sulphonamides.*
 - Diuretics: *Thiazides, Frusemide.*
2. Infection: *acute pyelonephritis.*
3. Immunological: *SLE.*
4. Irradiation.

II. Chronic interstitial nephritis:

1. Iatrogenic:
 - NSAIDs: prolonged use & abuse (analgesic nephropathy).
2. Infection: *chronic pyelonephritis & renal TB.*
3. Immunological: *SLE.*
4. Irradiation.
5. METABOLIC: *DM gout, nephrocalcinosis.*
6. TOXIC: *Lead, mercury.*
7. NEOPLASTIC: *MM.*
8. OBSTRUCTIVE UROPATHY.

CLINICAL PICTURE

1. Tubular abnormalities: *e.g. Fanconi syndrome, RTA.*
2. Anemia.
3. Proteinuria: *usually mild.*
4. RENAL FAILURE: *ARF or CRF.*
5. Features of the cause: *e.g. SLE.*

INVESTIGATIONS

1. Urine analysis:
 - *Proteinuria, hematuria, eosinophils.*
 - *Isosthenuria.*
3. Renal function tests:
 - Impaired: *if renal failure occurs.*
4. Renal biopsy:
 - It will show the pathology.
5. Investigations for the cause: *e.g. SLE.*

TREATMENT

1. Treatment of the cause: *e.g. SLE.*
2. Treatment of renal failure: *Dialysis or transplantation.*

TUBULAR NEPHROPATHIES

FANCONI SYNDROME

DEFINITION

- It is a disorder in which the proximal tubular function of the kidney is impaired, resulting in decreased reabsorption of electrolytes and nutrients back into the bloodstream.

ETIOLOGY

1. Hereditary

- Isolated.
- Wilson's disease.
- Cystinosis.

2. Acquired

- Multiple myeloma.
- Drugs: *expired tetracyclins.*

MANIFESTATIONS

1. Metabolic acidosis.
2. Hypokalemia.
3. Renal glycosuria.
4. Polyuria, polydipsia, dehydration.
5. Aminoaciduria.
6. Phosphaturia → rickets (in children) or osteomalacia (in adults).

TREATMENT

1. Fluid & electrolyte replacement.
2. TTT of the cause.

RENAL TUBULAR ACIDOSIS

DEFINITION

- It is a medical condition that involves an accumulation of acid in the body due to: failure of the kidneys to appropriately acidify the urine.

ETIOLOGY

1. Hereditary

- Isolated.
- Wilson's disease.
- Cystinosis.

2. Acquired

- Multiple myeloma.
- Drugs: *analgesic nephropathy, amphotericin B.*

TYPES

1. TYPE I: [Distal RTA]

- Metabolic acidosis.
- Hypokalemia.
- Nephrocalcinosis & renal stones.

2. TYPE II: [Proximal RTA]

- Isolated: *loss of HCO_3 only.*
- Part of Fanconi syndrome: *generalized proximal tubular dysfunction.*

TREATMENT

1. Fluid & electrolyte replacement.
2. TTT of the cause.

ACUTE PYELONEPHRITIS

ETIOLOGY

A. Causative organism:

1. Gram negative bacilli: 90 % of the cases
 - E. coli: 80 % of the cases.
 - Klebsiella, Proteus, Pseudomonas: 10 % of the cases.
2. Gram positive cocci: Staph. aureus, Strept. fecalis.
3. Others: RARE fungi, chlamydia, viruses.

B. Routes of infection:

1. Ascending infection: “the most common”
 - In cases of lower UTI.
2. Hematogenous route: “not common”
 - In cases of BACTEREMIA, e.g. SBE, Pneumonia
3. Lymphatic route.

C. Predisposing factors:

1. Impaired urine flow: “Urinary tract obstruction”
 - Renal pelvis: stone, tumour.
 - Ureters: stone, tumour, stricture.
 - Bladder: bladder neck obstruction, neurogenic bladder.
 - Urethra: stone, enlarged prostate, stricture.
2. Impaired immunity:
 - **A**IDS.
 - **B**ad general condition: general debility, old age, malnutrition.
 - **C**orticosteroids.
 - **D**iabetes mellitus.
3. Introduction of infection:
 - **C**atheterization, Cystoscopy, Surgery of the bladder.
 - **C**ystitis: “Honeymoon cystitis”.

PATHOLOGY

- ❖ Inflammation: of the mucous membrane of the kidney.
- ❖ Abscesses: Multiple small abscesses are present with streaks of pus.

COMPLICATIONS

1. Perinephric abscess.
2. Pyonephrosis.
3. Chronic pyelonephritis.
4. Septicemia.

CLINICAL PICTURE

Symptoms

General:

- Fever: acute with rigors.

Abdominal:

- Pain: acute pain in the loin radiating to the groin.
- Urinary symptoms: *frequency, dysuria, pyuria, strangury.*
- Nausea & vomiting.

Signs

General:

- Fever: acute with pallor.

Abdominal:

- Tenderness: in the renal angle.

Acute Necrotizing Papillitis

- A severe form of infection leading to papillary necrosis.
- It is most common in: Diabetes mellitus.
- It presents with severe acute pyelonephritis & severe hematuria.
- It is complicated by: ARF.

INVESTIGATIONS

1. Urinalysis:

- *Volume:* normal.
- *Aspect:* normal OR turbid.
- *pH:* ACIDIC in E. coli (most cases), ALKALINE in proteus.
- *Proteins:* mild proteinuria is present.
- *Microscopic examination:*
 - Cells: Many pus cells, Some RBCs, Organisms.
 - Casts: White cell casts.

2. Urine culture & sensitivity:

- Bacterial count more than 100,000 / ml: *denotes infection.*
- Bacterial count less than 100,000 / ml: *denotes contamination.*

3. Blood picture:

- Polymorphnuclear leucocytosis with shift to the left.

4. Renal function tests:

- Normal (except in ANP).

5. Renal imaging:

- e.g. PUT & ultrasonography, for detection of:
 - Predisposing factors: e.g. stones.
 - Complications: e.g. perinephric abscess.

DIFFERENTIAL DIAGNOSIS

1. Fever with rigors.
2. Abdominal pain.
3. Pyuria, e.g. cystitis.

TREATMENT**I. GENERAL TREATMENT**

- Rest in bed.
- Excessive fluid intake.
- Analgesics & antipyretics.

II. SPECIFIC TREATMENT**1. Antibiotics:***"for 1 – 2 weeks"*

- Before the results of urine C & S: give a broad spectrum antibiotic, e.g.
 - Quinolones: *Ciprofloxacin* 500 mg bid orally.
 - Tetracyclines: *Doxycycline* 100 mg bid orally.
- Resistant & Recurrent cases: give antibiotic according to urine C & S:
 - For E. coli: *Ciprofloxacin* 500 mg bid orally, or *Nitrofurantoin* 100 mg / 8 h.
 - For Pseudomonas: *Carbimicillin* 1 gm / 6 h IV.
 - For other Gm– ve bacilli: *Gentamycin* 80 mg / 8 h IV or IM.

2. Change the pH of the urine:

- If the urine is acidic: give *sodium bicarbonate*.
- If the urine is alkaline: give *ascorbic acid*.

III. CORRECTION OF PREDISPOSING FACTORS.**IV. TREATMENT OF COMPLICATIONS.**

CHRONIC PYELONEPHRITIS

DEFINITION

- CHRONIC interstitial nephritis resulting from RECURRENT bacterial infection of the kidney.
- The disease is either: *unilateral* or *bilateral asymmetrical*.

ETIOLOGY

- RECURRENT acute pyelonephritis.

PATHOLOGY

1. Macroscopic appearance:

- Kidney: SMALL with *irregular surface* & *adherent capsule*.

2. Microscopic appearance:

- Tubules: atrophic.
- Glomeruli: periglomerular fibrosis.
- Interstitial tissue: chronic inflammatory cells & excessive fibrosis.

CLINICAL PICTURE

- The patient may present with *one* OR *more* of the following manifestations:
 1. **Recurrent** attacks of acute pyelonephritis.
 2. **Recurrent** attacks of general non-specific symptoms, e.g. *back pain*, *fatigue*, *anorexia*.
 3. **Chronic** anemia.
 4. **Chronic** renal failure.
 5. **Secondary** hypertension.
 6. **Symptomless** proteinuria.

INVESTIGATIONS

1. Urinalysis:

- *Proteins:* mild proteinuria is present (less than 3.5 gm / 24 hours).
- *Microscopic examination:*
 - Cells: Pus cells, RBCs, Organisms.
 - Casts: **White cell casts.**
- *In late stages:* features similar to CRF.

2. Urine culture & sensitivity:

- Bacterial count more than 100,000 / ml: *denotes infection.*
- Bacterial count less than 100,000 / ml: *denotes contamination.*

3. Blood picture:

- Polymorphnuclear leucocytosis.
- Anemia.

4. Renal function tests:

- Impaired in late cases.
- Impaired tubular functions more than glomerular functions.
- Impaired functions of one kidney more than the other kidney.

5. Renal imaging:

- e.g. PUT & ultrasonography & IVP, for detection of:
 - Predisposing factors: e.g. stones.
 - SMALL-Sized kidneys: usually unequal.

6. Renal biopsy:

- It will show the pathology.

TREATMENT

I. GENERAL TREATMENT

- Symptomatic treatment for: *back pain, fatigue, anorexia.*

II. SPECIFIC TREATMENT " ANTIBIOTICS "

- **D**rugs: according to urine C & S.
- **D**oses: reduced doses because of impaired excretion of the drugs.
- **D**uration: prolonged & repeated courses of ttt may be needed.

III. CORRECTION OF PREDISPOSING FACTORS.

IV. TREATMENT OF COMPLICATIONS, e.g. CRF, Hypertension.

V. UNILATERAL NEPHRECTOMY

- In advanced unilateral cases with complete loss of function.

RENAL TUBERCULOSIS

ETIOLOGY

- **Organism:** Mycobacterium TB.
- **Infection:** Hematogenous spread from a primary infection.
- **Predisposing factor:** Immunodeficiency, e.g. AIDS.

CLINICAL PICTURE

Symptoms

General:

- TB toxemia: *night sweats, night fever, loss of weight, loss of appetite.*

Abdominal:

- Pain: *pain in the loin radiating to the groin.*
- Urinary symptoms: *frequency, dysuria, pyuria, strangury, hematuria.*

Signs

General:

- Fever: *with pallor, toxic face, loss of weight.*

Abdominal:

- Tenderness: *in the renal angle.*

Patients may have active TB in other parts of body.

COMPLICATIONS

1. **C**RF: *in bilateral cases.*
2. **C**alcification.
3. **S**secondary hypertension.
4. **S**secondary amyloidosis.
5. **S**pread: *e.g. genital TB.*
6. **S**tricture of the ureter: *leading to obstructive uropathy.*

INVESTIGATIONS

1. Urinalysis:

- Sterile pyuria.
- Hematuria.
- Urine is subjected to:
 - Staining: using ZN stain.
 - C & S: using Lowenstein medium & BACTEC.
 - Animal inoculation: using guinea pig.

2. Blood investigations:

- Anemia.
- ESR: *markedly elevated.*

3. Renal imaging:

PUT & ultrasonography & IVP, for detection of:

- *Calcification.*
- *Stricture of the ureter.*

4. Renal biopsy:

- It will show the pathology: refer to “Chest”.

5. PCR: (Polymerase Chain Reaction)

- For detection of mycobacterial DNA.

6. Tuberculin test:

- It is usually positive.

TREATMENT

I. GENERAL TREATMENT

- *Rest in bed.*
- *Proper nutrition.*

II. SPECIFIC TREATMENT

- Anti – TB ttt: refer to “Chest”.

III. TREATMENT OF COMPLICATIONS

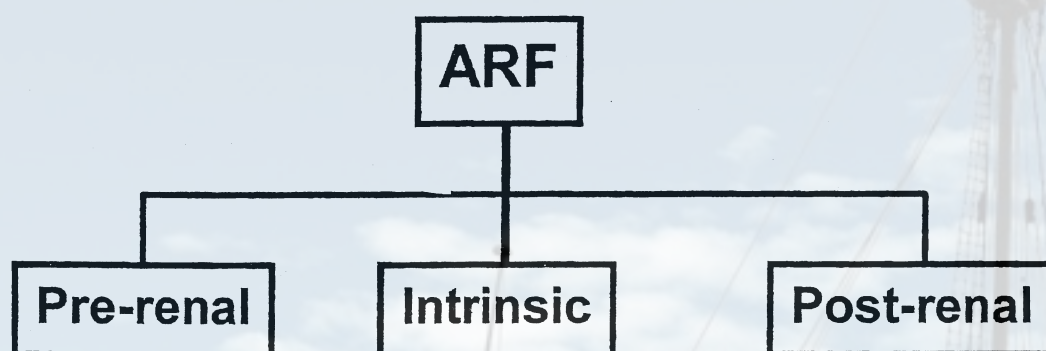
- Surgery: for stricture of the ureter.

ACUTE RENAL FAILURE

DEFINITION

- A disease characterized by acute rapid decline of the renal functions:
 - It is usually associated with oliguria: urine volume less than 400 ml / day.
 - It is frequently reversible.

ETIOLOGY



I. PRE-RENAL

55 %

"Renal Hypoperfusion"

A. Decrease in blood volume:

1. SEVERE HEMORRHAGE.
2. SEVERE HYPOALBUMINEMIA.
3. Skin *fluid loss*: Burns, Excessive sweating.
4. GIT *fluid loss*: Vomiting, Diarrhea.
5. Renal *fluid loss*: Diuretics, Osmotic diuresis (e.g. DKA), Polyuria.

B. Decrease in the renal blood flow:

1. **S**hock: cardiogenic shock & septic shock.
2. **S**evere renal ischemia: bilateral renal artery *thrombosis* or *embolism*.
3. **S**evere heart *failure*: & other causes of ↓ CO as *sustained arrhythmias*.
4. **S**evere liver *failure*: hepatorenal syndrome.

II. INTRINSIC RENAL 40 % “Diseased renal parenchyma”

A. Diseases of the tubules:

1. Acute tubular necrosis (ATN): “the most common cause”

a. Ischemic:

- Prolonged reduction of the renal blood flow, ie:
Persistence of the pre – renal causes (any cause).

b. Toxic:

- Drugs: *Aminoglycosides, cyclosporine.*
- Heavy metals: *mercury, lead.*
- RADIO CONTRAST AGENTS.

2. Acute necrotizing papillitis.

3. Acute interstitial nephritis.

4. Tubular obstruction:

a. Crystals:

- Uric acid: in gout.

b. Proteins:

- Hemoglobin: in hemolytic crisis.
- Myoglobin: in *rhabdomyolysis* *
- Myeloma proteins: in multiple myeloma.

B. Diseases of the glomeruli: “GN”

1. Membrano – proliferative GN.
2. Mesangio – proliferative GN.
3. Diffuse proliferative GN → acute GN.
4. Crescentic GN → RP GN e.g. GPS.

* Mention the causes of rhabdomyolysis

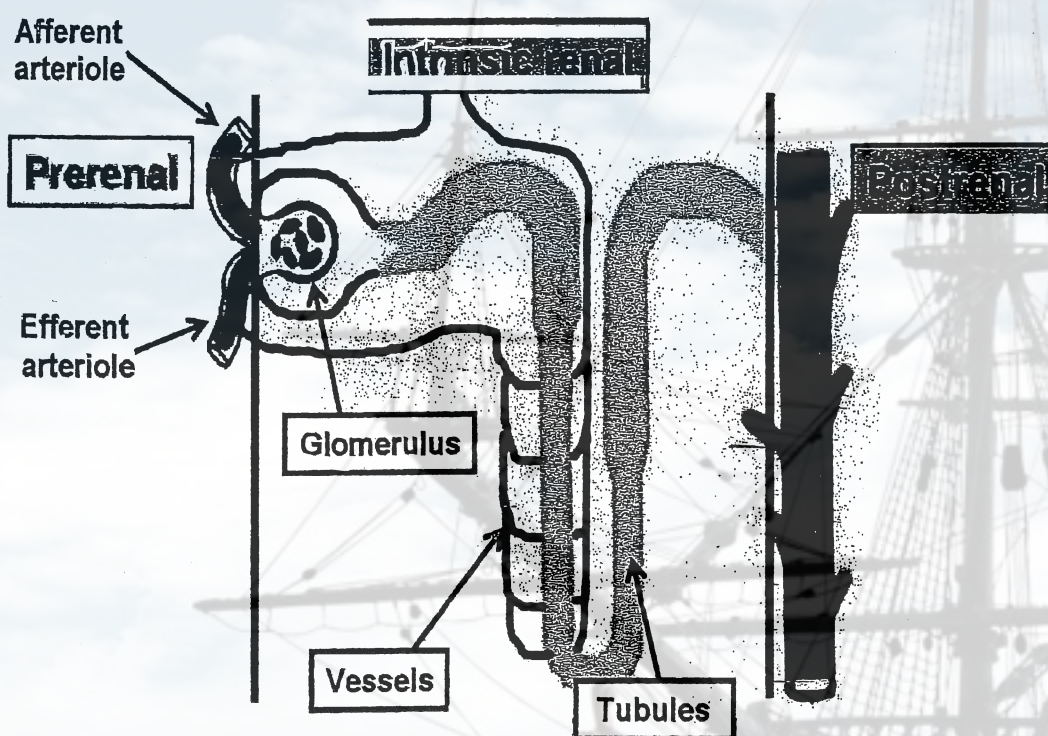
C. Vascular diseases:

1. Malignant hypertension.
2. Eclampsia.
3. Vasculitis: e.g. SLE, PAN.
4. MAHA: HUS, TTP, DIC.

III. POST-RENAL

5 % "Obstruction of the urine flow"

1. Bilateral ureteric obstruction: e.g. stones.
2. Unilateral ureteric obstruction: in a single kidney.
3. Obstruction of the urethra or the bladder neck.



Pathogenesis of ATN

- ATN passes through 3 phases:

1. Oliguric phase: [1 – 2 weeks] *“Injury of the tubules with ↓ GFR due to”*

- LEAK: of the glomerular filtrate through the injured tubules back to the blood.
- Intrarenal VC: due to release of VC mediators from injured endothelial cells.
- Obstruction: of the tubules by the injured tubular debris.
- All the above : will result in decline of GFR → **decreased urine volume.**

2. Diuretic phase: *“partial recovery of the tubules with excessive diuresis due to”*

- Excretion of retained salt & water.
- Delayed recovery of tubular reabsorption compared to glomerular filtration.
- All the above: will result in **increased urine volume.**

3. Recovery phase: *“complete recovery of the tubules”*

- The completely recovering tubules will regain their power to fully reabsorb water and to concentrate the urine, leading to **normal urine volume.**

Pathology of ATN

a. Ischemic:

- Distribution: Patchy necrosis of the whole tubule.
- Basement membrane: Damaged.

b. Toxic:

- Distribution: Diffuse necrosis of the proximal tubule.
- Basement membrane: Intact.

CLINICAL PICTURE

I. Manifestations of the oliguric phase

1. Oliguria: *"few days to few weeks"*
 - Volume:
 - Less than 400 ml of urine per day.
 - Absolute anuria is more common in post-renal obstruction.
2. Hypervolemia: *"Overhydration"*
 - Circulation:
 - Congestion (neck veins, pulmonary).
 - Oedema (systemic, pulmonary).
 - HYPERTENSION.
 - Brain:
 - Convulsions, Confusion, Coma.
3. Hyperkalemia: *"see later"*.
4. Acidosis: *"see later"*
5. Uremic syndrome: *"Clinical features of uremia *"*
 - A** Anorexia, nausea, vomiting, hiccough.
Anemia.
Astrexia.
 - B** Bleeding tendency, GIT Bleeding.
Pruritus, Pericarditis, Pleurisy.
 - C** Convulsions, Confusion, Coma.

II. Manifestations of the diuretic phase

- Volume: urine volume reaches up to 5 – 10 L / day.
- Effect: hypotension, dehydration, electrolyte loss, e.g. Na, K, Mg.

III. Manifestations of the recovery phase

- Volume: urine volume returns to normal.
- Effect: clinical manifestations improve.

* What is meant by uremia ??

INVESTIGATIONS

1. Urinalysis:

- *Volume:* Less than 400 ml / day, may be absolute anuria.
- *Aspect:* Dark.
- *Specific gravity:* Low fixed “in the range of 1010” in cases of ATN.
- *Proteins:* mild proteinuria is present.
- *Microscopic examination:*
 - Cells: Epithelial cells & RBCs.
 - Casts: **Granular casts.**

2. Blood investigations:

- ANEMIA.
- Increased: K, P.
- Decreased: Na, Ca, HCO₃, pH.

3. Renal function tests:

- There is marked impairment of the renal functions:
 - Increased: creatinine, urea, uric acid.
 - Decreased: creatinine clearance, an early good estimate of ↓ GFR.
 - **FE Na:** < 1 % = pre – renal failure, > 1 % = ATN.

4. Renal imaging: “PUT, Ultrasonography, CT, MRI”

- Post-renal obstruction: e.g. stones.

5. Renal biopsy: indicated in:

- Persistent ARF: more than 4 weeks.
- Unexplained ARF.

6. ECG: may show features of Hyperkalemia (see later).

7. Investigations for the cause.

RIFLE¹ criteria for the diagnosis of ARF

	GFR Criteria	Urine Output Criteria	
Risk	Increased SCreat x1.5 or GFR decrease > 25%	UO < 5ml/kg/h x 6hr	R
Injury	Increased SCreat x2 or GFR decrease > 50%	UO < 5ml/kg/h x 12hr	I
Failure	Increase SCreat x3 GFR decrease 75% OR SCreat ≥ 4mg/dl	UO < 3ml/kg/h x 24hr or Anuria x 12 hrs	F
Loss	Persistent ARF = complete loss of kidney function > 4 weeks		L
ESKD	End Stage Kidney Disease (≥ 3 months)		E

- The classification system includes separate criteria for: Serum creatinine (SCreat) and Urine output (UO).
- A patient can fulfill the criteria through changes in: SCreat or changes in UO, or both.
- The criteria that lead to the worst possible classification should be used.
- **RIFLE**:
 - **R**isk of renal dysfunction
 - **I**njury to the kidney
 - **F**ailure of kidney function
 - **L**oss of kidney function
 - **E**nd stage kidney disease

¹ **Ref:** Bellomo *et al.* (2004). "Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group". *Crit Care* 8 (4): R204-12. doi:10.1186/cc2872

TREATMENT

1. Exclusion

- **Exclusion of urinary retention:** by bladder catheterization.
- **Exclusion of post-renal obstruction:** by investigations, & obstruction should be treated.

2. Treatment of the cause

- Pre-renal causes: IV fluids for hypovolemia, or blood transfusion for hemorrhage.
- Toxic agents: should be stopped.

3. Induction of diuresis

“treatment of the oliguric phase”

- Frusemide: 2 – 10 mg / Kg IV.
- Dopamine: 2 ug / Kg / min. IV infusion.
- Mannitol: 500 ml of 25 % solution IV infusion.

4. Conservative treatment

a) Diet treatment:

- Salt & fluid restriction: to correct Hypervolemia.
- Potassium restriction: to correct Hyperkalemia.
- Protein restriction: to lower the blood urea (0.5 gm / Kg / day).
- Carbohydrates: allowed in excess to decrease protein catabolism.
- Fats: avoided because they are nauseating.

b) Treatment of Hypervolemia:

- Salt & fluid restriction.
- Monitoring: CVP, BP, Oedema, Urine output.

c) Treatment of Hyperkalemia:

- See later.

d) Treatment of acidosis:

- Sodium bicarbonate: by IV infusion in severe cases (pH less than 7.2).

e) Symptomatic treatment:

- Hypertension, pulmonary oedema, anemia, vomiting, etc.....
- Hyperphosphatemia: dietary restriction + P binders, Calcium supplementation.

f) Treatment during the diuretic phase:

- Adequate fluid intake to avoid hypovolemia & dehydration.
- Adequate replacement of electrolyte loss, e.g. Na, K, Mg.

5. Dialysis treatment

- Indications

A) Laboratory:

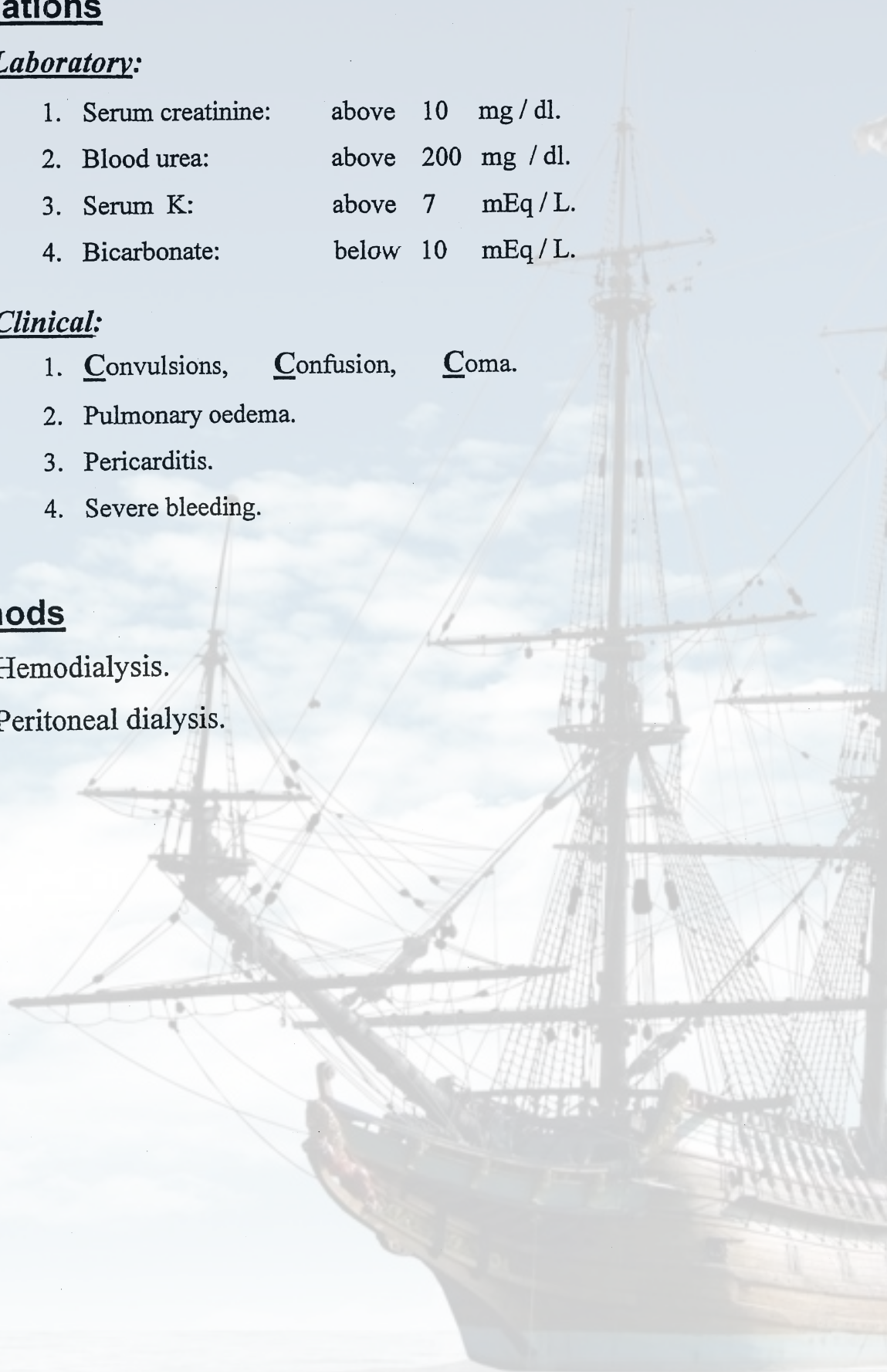
1. Serum creatinine: above 10 mg / dl.
2. Blood urea: above 200 mg / dl.
3. Serum K: above 7 mEq / L.
4. Bicarbonate: below 10 mEq / L.

B) Clinical:

1. Convulsions, Confusion, Coma.
2. Pulmonary oedema.
3. Pericarditis.
4. Severe bleeding.

- Methods

1. Hemodialysis.
2. Peritoneal dialysis.



CHRONIC RENAL FAILURE (CRF)

DEFINITION

- A disease characterized by gradual progressive deterioration of renal functions over months or years:
 - It is IRREVERSIBLE.
 - The clinical manifestations of end-stage renal failure are: “uremia or uremic syndrome”.

ETIOLOGY

[the most common cause is DM]

1. INFECTIONS

- Chronic Pyelonephritis.
- Bilateral renal TB.

2. IMMUNE

- Chronic GN.
- *SLE, PAN, HSP, GPS, EMC.*
- Scleroderma.

3. METABOLIC

- Diabetic *nephropathy*, ♣ *gouty nephropathy.*
- Amyloidosis, ♣ *nephrocalcinosis.*

4. MALIGNANT

- Multiple myeloma.

5. TOXIC

- Iatrogenic: *analgesic nephropathy, irradiation nephropathy.*
- Heavy metals: *lead, mercury.*

6. VASCULAR

- Hypertension: *especially malignant hypertension.*
- Recurrent renal infarctions, *e.g. sickle cell anemia.*

7. OBSTRUCTIVE

- Bilateral hydronephrosis. ♣

8. CONGENITAL

- Polycystic kidney. ♣
- Renal hypoplasia.

PATHOLOGY

1. Macroscopic appearance:

- Kidney: small with *irregular surface & adherent capsule*.

2. Microscopic appearance:

- Interstitial tissue: excessive fibrosis.
- Tubules: atrophic.
- Glomeruli: atrophic.

CLINICAL PICTURE

The uremic syndrome results from both: glomerular & tubular dysfunctions:

1. WATER, ELECTROLYTE & ACID-BASE DISORDERS

1) Water:

- Polyuria: due to:
 - Osmotic diuresis: due to increased solute load per nephron.
 - Failure of tubules to respond to ADH.
- Oliguria:
 - May occur in late stages.
- Isothenuria:
 - Low fixed specific gravity "in the range of 1010".
- Decreased ability to concentrate or dilute urine:
 - Increased water intake → hypervolemia & overhydration.
 - Decreased water intake → hypovolemia & dehydration.

2) Sodium: 2 types

- Sodium retention:
 - It occurs in most patients especially in glomerular diseases.
 - Cause: it is due to inability of the kidney to excrete Na load.
 - Effect: hypervolemia, hypertension, pulmonary congestion, oedema.
- Sodium loss: "salt – losing nephropathy"
 - It occurs more commonly in patients with tubular diseases.
 - Cause: it is due to inability of the tubules to reabsorb Na.
 - Effect: hypotension & muscle cramps.

3) Potassium:

- Increased serum K due to extracellular shift of K and due to ??

4) Phosphorus & Calcium:

- Increased P & decreased Ca.

5) Metabolic acidosis:

- Due to failure to secrete H & to form HCO₃.

2. CARDIOVASCULAR MANIFESTATIONS

- 1) Hypertension: due to salt & water retention or increased rennin.
- 2) Arterial disease: accelerated atherosclerosis due to hyperlipidemia.
- 3) Pericarditis: dry or hemorrhagic.

3. RESPIRATORY MANIFESTATIONS

- 1) **R**apid deep breathing: Kussmaul's breathing, due to acidosis.
- 2) **R**ecurrent chest infections: due to impaired immunity.
- 3) **P**ulmonary oedema: due to hypervolemia.
- 4) **P**leurisy.
- 5) **U**remic asthma.
- 6) **U**lcers: in the respiratory tract → hemoptysis.

4. GIT MANIFESTATIONS

- 1) Mouth: ammoniacal odour + stomatitis.
- 2) Tongue: dry + coated.
- 3) Stomach: anorexia + nausea + vomiting + hiccough + hematemesis.
- 4) Intestine: constipation + bleeding.
- 5) Uremic dysentery: diarrhoea + tenesmus with blood & mucus in stools due to: ulcers in the mucus membrane of the colon & rectum.
- 6) Increased incidence of HCV in patients on regular hemodialysis.

5. RENAL MANIFESTATIONS

- See before: Water disorders.

6. NEUROLOGICAL MANIFESTATIONS

- 1) **A**pathy.
- 2) **A**sterixis.
- 3) **P**sychiatric symptoms: irritability, depression.
- 4) **P**eripheral neuropathy: & myopathy.
- 5) **C**onvulsions.
- 6) **C**onfusion & coma: uremic coma or uremic encephalopathy.
- 7) **D**ialysis dementia: usually due to aluminium intoxication.
- 8) **D**rowsiness, fatigue, headache & lack of concentration.

7. GENITAL MANIFESTATIONS

- Males: impotence & infertility.
- Females: amenorrhea & infertility.

8. MUSCULOSKELETAL MANIFESTATIONS

- 1) Renal osteodystrophy: "due to secondary hyperparathyroidism"
 - *Osteomalacia, osteoporosis, osteitis fibrosa cystica.*
 - Bone pains & pathological fractures may occur.
- 2) Myopathy.
- 3) Muscle cramps: due to decreased Na.



Osteitis fibrosa cystica

9. SKIN MANIFESTATIONS

- **P**allor: & earthy colour of the skin
- **P**ruritus.
- **P**igmentation.
- Uremic frost: deposits of urea crystals.
- Dehydrated skin.

10. BLOOD MANIFESTATIONS

1) Anemia:

- ↓ production of EPO.
- ↓ iron.
- ↓ vitamins (folic acid, vitamin B₁₂).
- ↓ life span of RBCs (hemolysis).
- HYPERPARATHYROIDISM.

2) Bleeding tendency: due to:

- Platelet dysfunction: *“the most important factor”*
- Toxic inhibition of the clotting factors.

3) Increased liability to infections: due to:

- Lymphocyte & phagocyte dysfunction: *“the most important factor”*
- Increased exposure to infection in patients on dialysis.

11. EYE MANIFESTATIONS

- Uremic retinitis.

12. METABOLIC MANIFESTATIONS

- Disturbance in CHO metabolism may be *one of 2 types*:
 - Early: Hypoglycemia due to decreased renal clearance of insulin.
 - Later: Hyperglycemia (IGT or secondary diabetes) due to insulin resistance.

13. MANIFESTATIONS DUE TO DIALYSIS COMPLICATIONS

- See later.

INVESTIGATIONS

1. Urinalysis:

- *Volume:* Polyuria, about 3 – 4 L / day, Oliguria occurs in late stages.
- *Aspect:* Pale.
- *Specific gravity:* Low fixed “in the range of 1010”.
- *Proteins:* mild proteinuria is present.
- *Microscopic examination:*
 - Casts: **Broad casts** & granular casts.

2. Blood investigations:

- ANEMIA.
- Increased: K, P.
- Decreased: Ca, HCO₃, pH.
- Variable: Na.

3. Renal function tests:

- There is marked impairment of the renal functions:
 - Increased: creatinine, urea, uric acid.
 - Decreased: creatinine clearance, an early good estimate of ↓ GFR.

4. Renal imaging: “PUT, Ultrasonography, CT, MRI”

- The cause.
- The renal size.

5. Renal biopsy:

- Will reveal the pathology.

6. ECG: may show features of Hyperkalemia (see later).

7. Investigations for the cause.

TREATMENT

I. DIET TREATMENT

1. Fluids:

- Fluid intake depends on urine output & other losses.

2. Salt:

- Restricted in sodium retention, hypertension or heart failure.
- Increased in salt – losing nephropathy.

3. Potassium:

- Restricted to correct hyperkalemia.

4. Proteins:

- Restricted to lower the blood urea (0.5 gm / Kg / day).

5. Fats:

- Restricted to correct hyperlipidemia & because they are nauseating.

6. Carbohydrates:

- Allowed in excess to decrease protein catabolism.

II. SYMPTOMATIC TREATMENT

- Anemia: EPO, iron & folic acid, packed red cell transfusion.
- Renal osteodystrophy: Calcium, vitamin D, + ttt of hyperphosphatemia.
- Hypertension, heart failure, infections, vomiting, convulsions, pruritus etc....

III. TREATMENT OF THE CAUSE

- e.g. Adequate control of DM.
- e.g. Prevention of Chronic Pyelonephritis.

IV. TREATMENT OF ESKD

1. Dialysis treatment

• Indications

A) Laboratory:

1. Serum creatinine: above 10 mg / dl.
2. Creatinine clearance: below 10 ml / min.

B) Clinical:

- Conservative ttt becomes inadequate to control the clinical symptoms.

• Methods

1. Hemodialysis.
2. Peritoneal dialysis.

• Complications

1. Hemodialysis: Dialysis dementia, air embolism.
2. Peritoneal dialysis: Peritonitis, abdominal hernias.
3. Both types: HCV, AIDS & other infections.

2. Renal transplantation

- It is the ONLY curative ttt for irreversible renal failure.

-
- **Important question:** **“Self – assessment”**
“Mention the classification of CKD according to the severity.”

HYPERKALEMIA

ETIOLOGY

1. INCREASED INTAKE

“Iatrogenic”

- Excessive therapy with IV potassium.
- Excessive transfusion of stored blood.

2. DECREASED LOSS

- Renal failure: *Acute & Chronic.*
- Adrenal failure: *Addison’s disease.*
- Drugs: *Potassium – sparing diuretic (e.g. spironolactone),
ACE inhibitor (e.g. captopril).*

3. EXTRACELLULAR SHIFT

- Acidosis.
- Insulin deficiency.
- Tissue damage (*lysis*): *hemo*lysis, *rhabdomyo*lysis, *tumour* lysis syndrome.

CLINICAL PICTURE

- | | | | |
|------------|--------------|------------------|-----------------------------|
| 1. Heart: | Bradycardia, | Heart block, | Cardiac arrest in diastole. |
| 2. Muscle: | Myopathy, | Muscle weakness, | Muscle paralysis. |

INVESTIGATIONS

1. Serum K:

- Increased (normal: 3.5 – 5 mEq / L).

2. ECG:

- P-R interval: prolonged.
- QRS complex: widened.
- T wave: tall & peaked (hyperacute T wave).
- Bradycardia, Heart block.

3. Investigations for the cause:

- e.g. elevated urea & creatinine in acute & chronic renal failure.

TREATMENT

1. DECREASED INTAKE

- Diet: restriction of potassium in diet.
- Drugs: restriction of drugs that ↑ K, e.g. *spironolactone* & *captopril*.
- Decrease absorption: resins, as cation-exchange resins.

2. INCREASED LOSS

- Diuretics: e.g. frusemide.
- Dialysis: in severe cases (K above 7 mEq/L).

3. INTRACELLULAR SHIFT

- Correction of acidosis: NaHCO₃ 100 mEq IV.
- Insulin + glucose: Regular insulin 10 U + glucose 25 gm IV infusion.

4. ANTAGONIZE THE EFFECT OF HYPERKALEMIA ON HEART

- Calcium gluconate: 10 ml of 10 % solution over 10 minutes.

HYPOKALEMIA

ETIOLOGY

1. DECREASED INTAKE

- IV fluid therapy: deficient in potassium.
- Diet: deficient in potassium.
- Decreased absorption: malabsorption syndrome.

2. INCREASED LOSS

- Renal loss:
 - Diuresis: Non potassium-sparing diuretics (loop & thiazides), osmotic diuresis.
 - Cushing's syndrome & Conn's syndrome & Corticosteroid therapy.
- GIT loss:
 - Diarrhoea: especially secretory diarrhoea.
 - Vomiting.
- Excessive tapping of ascites.

3. INTRACELLULAR SHIFT

- Alkalosis.
- Insulin therapy.
- Diet rich in carbohydrates.

CLINICAL PICTURE

1. Heart: Arrhythmias, especially in patients receiving digitalis.
2. Muscle: Myopathy, Muscle weakness, Muscle paralysis.
3. GIT: Constipation, distension, paralytic ileus.
4. Kidney: Damage of the renal tubules.
5. CNS: Tetany due to associated *alkalosis*.

INVESTIGATIONS

1. Serum K:
 - Decreased (normal: 3.5 – 5 mEq / L).
2. ECG:
 - ST segment: depressed.
 - T wave: flat or inverted.
 - Prominent U wave.
3. Investigations for the cause:
 - e.g. investigations for malabsorption syndrome.

TREATMENT

1. Potassium replacement:
 - In severe cases: IV potassium with careful monitoring.
 - In mild cases: oral potassium is preferred to avoid hyperkalemia.
2. Treatment of the cause.

METABOILIC ACIDOSIS

ETIOLOGY

1. Increased acid:

a) Increased acid production:

- *Endogenous:* Diabetic ketoacidosis, Lactic acidosis.
- *Exogenous:* Salicylates, Methyl alcohol.

b) Decreased acid excretion:

- *Renal failure:* Acute & Chronic.

2. Decreased alkali:

a) GIT causes:

- *Severe diarrhoea.*
- *Fistula:* pancreatic, biliary, intestinal.

b) Renal causes:

- *Tubular defects:* Fanconi syndrome, Renal tubular acidosis.
- *Drugs:* Spironolotone.

CLINICAL PICTURE

1. *Respiratory:* Hyperventilation: Kussmaul's breathing (rapid deep breathing).
2. *Heart:* Myocardial depression.
3. *Bone:* Decalcification in chronic cases.
4. *CNS:* Drowsiness & impaired consciousness in severe cases.

INVESTIGATIONS

1. Low pH of the blood.
2. Low HCO_3^- & PCO_2 .
3. Investigations for the cause.

TREATMENT

1. NaHCO_3 IV.
2. Treatment of the cause.

RESPIRATORY ACIDOSIS

ETIOLOGY

- All causes of hypercapnic respiratory failure that occur due to hypoventilation: *"Refer to chest"*.

CLINICAL PICTURE

- Features of respiratory failure: *"Refer to chest"*.

INVESTIGATIONS

1. Low pH of the blood.
2. High PCO_2 & HCO_3 .
3. Investigations for the cause.

TREATMENT

- Treatment of respiratory failure: *"Refer to chest"*.

METABOLIC ALKALOSIS

ETIOLOGY

1. Increased intake of alkali:

- Excessive ttt with bicarbonate.
- Milk-alkali syndrome in ttt of peptic ulcer.

2. Decreased acid:

- a) GIT loss: Vomiting & gastric drainage.
- b) Renal loss: Diuretics esp. loop diuretics, Conn's syndrome, Cushing's syndrome.

3. Hypokalemia.

CLINICAL PICTURE

1. *Respiratory:* Hypoventilation: shallow slow breathing.
2. *Tetany.*
3. *Manifestations of hypokalemia may occur.*

INVESTIGATIONS

1. High pH of the blood.
2. High HCO_3^- & PCO_2
3. Investigations for the cause.

TREATMENT

1. Treatment of the cause.
2. Diluted HCL: in severe cases.

RESPIRATORY ALKALOSIS

ETIOLOGY

ALL CAUSES OF HYPERVENTILATION: e.g.

- Hysteria.
- Head injury or encephalitis.
- Heat stroke.
- Heart failure or pulmonary embolism or pneumonia.

CLINICAL PICTURE

1. Hyperventilation.
2. Parasthesia.
3. Tetany: in severe cases.

INVESTIGATIONS

1. High pH of the blood.
2. Low PCO_2 & HCO_3^- .
3. Investigations for the cause.

TREATMENT

1. Treatment of the cause.
2. Hysterical cases: should rebreath in a paper bag + sedation.

PROTEINURIA

DEFINITION

- Presence of more than 300 mg of proteins in 24-hour urine.

ETIOLOGY

1. Glomerular proteinuria:

- Pathogenesis: increased glomerular permeability.
- Causes: nephritic syndrome & nephrotic syndrome.
- Amount: nephritic syndrome: $< 3.5 \text{ gm} / 24 \text{ h}$, nephrotic syndrome: $> 3.5 \text{ gm} / 24 \text{ h}$.

2. Tubular proteinuria:

- Pathogenesis: decreased reabsorption of the normally filtered proteins.
- Causes: tubular diseases & interstitial nephritis.
- Amount: mild proteinuria: $< 2 \text{ gm} / 24 \text{ h}$.

3. Overflow proteinuria:

- Pathogenesis: increased filtration of proteins of low molecular weight.
- Causes: hemoglobinuria, myoglobinuria, Bence-Jones proteinuria.
- Amount: variable ranging from: $0.2 - 10 \text{ gm} / \text{day}$.

4. Miscellaneous causes:

- Pathogenesis: not clear.
- Causes: HF, Heavy exercise, High fever, Anemia, Convulsions.
- Amount: mild proteinuria: $< 2 \text{ gm} / 24 \text{ h}$.

NB False proteinuria

- It results from loss of protein from the lining of the UT, e.g. INFECTIONS.

MICROALBUMINURIA

- Pathogenesis: excretion of small amounts of albumin in urine due to nephropathy.
- Causes: early indicator of diabetic nephropathy.
- Amount: minimal proteinuria: $150 - 300 \text{ mg} / 24 \text{ h}$.